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MEDICAL PROCEEDINGS

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A Mould Active Against Polio Viruses
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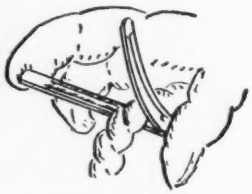

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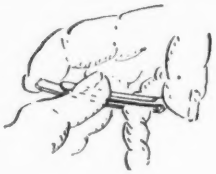

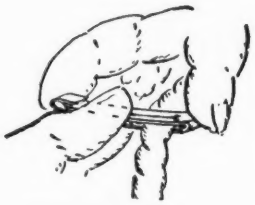

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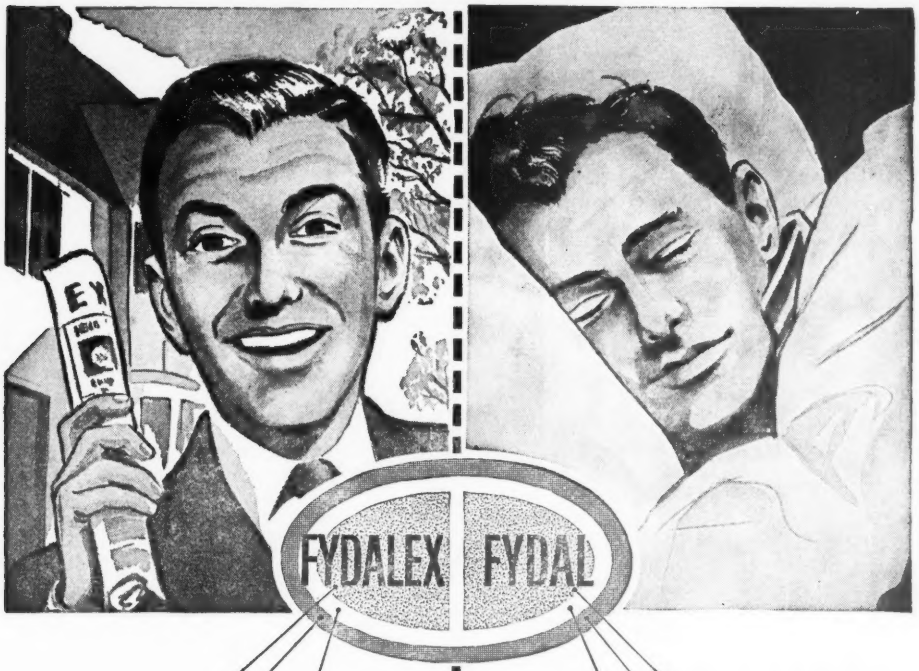

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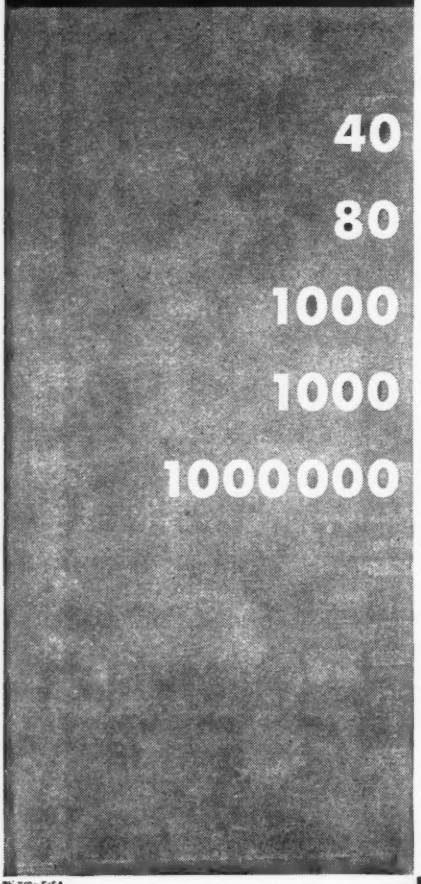

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Editor : Redakteur

H. A. Shapiro, B.A., Ph.D., M.B., Ch.B., F.R.S.S.Af.

Vol. 5 | 26 December 1959 Desember 26 | No. 26

SEASONAL GREETINGS

We take this opportunity of wishing all our colleagues a very merry Christmas and a happy New Year.

SEISOENSGROETE

Ons wil al ons kollegas 'n baie geseënde Kersfees en 'n gelukkige Nuwejaar toewens.

EDITORIAL · REDAKSIONEEL

A MOULD ACTIVE AGAINST POLIO VIRUSES

An interesting report has recently appeared on a new anti-viral agent.[1] This is known as helenine and is produced by *Penicillium funiculosum* using conventional fermentation procedures.

The compound is found only in the mycelium and it is not exuded by the mould into the nutrient medium. It has recently been identified and purified and is said to be a ribonucleoprotein, which is in the same class of compound as the virus it attacks.

The structure of helenine is not yet definite and attempts are being made to improve its stability. It is stated that the dissociation of helenine is not oxidative but due to weakness of the molecular structure. Helenine cannot be subjected to freezing or thawing hence it may be better, it is considered, to modify the present buffered aqueous solution in which it is dissolved.

It appears that helenine is the first nucleoprotein to be found effective against viruses and preliminary investigations have shown that

1. Lewis, U. J. (1959): Chemical Age, **82**, 388.

'N SKIMMEL WAT AKTIEF TEEN POLIO-VIRUSSE OPTREE

'n Interessante verslag oor 'n nuwe virusbestrydende middel[1] het onlangs verskyn. Hierdie middel staan bekend as helenien, en dit word geproduseer deur *Penicillium funiculosum* aan die gewone gistingsprosesse te onderwerp.

Die verbinding word alleen in die skimmeldraad aangetref, en word nie deur die skimmel in die voedingsmedium uitgesyfer nie. Dit is onlangs geïdentifiseer en gesuiwer, en daar word beweer dat dit 'n ribonukleoproteïen is wat onder dieselfde tipe verbinding ressorteer as die virus wat daardeur aangeval word.

Die struktuur van helenien is nog nie definitief bepaal nie, en pogings word aangewend om die stabiliteit daarvan te verbeter. Daar word verklaar dat die dissosiasie van helenien nie oksidatief is nie, maar wel aan 'n swakheid van die molekuulstruktuur toegeskryf moet word. Helenien kan nie aan bevriesing of ontdooiing onderwerp word nie, en gevolglik word daar gemeen dat dit miskien beter sal wees om die huidige gebufferde waterige middel waarin dit opgelos is, te wysig.

Dit skyn dat helenien die eerste nukleoproteïen is wat doeltreffend teen virusse optree, en voorlopige

1. Lewis, U. J. (1959): Chemical Age, **82**. 388.

the agent can prevent the development of poliomyelitis in monkeys when administered before symptoms appear. Mice are said to be protected against a form of encephalitis and some other viruses.

navorsingswerk het reeds aangetoon dat die middel die ontwikkeling van poliomiëlitis by bobbejane voorkom as dit toegedien word voordat die simptome hul verskyning maak. Daar word ook beweer dat dit muise vrywaar teen 'n bepaalde vorm van harsingsontsteking en sommige ander virusse.

DIE BEHANDELING VAN KORONÊRE SLAGAARSIEKTE MET PARENTERALE MAGNESIUMSULFAAT

In 'n vorige uitgawe[8] het ons 'n referaat uit Tasmanië gepubliseer oor die behandeling van koronêre slagaarsiekte met parenterale magnesiumsulfaat. Die Tasmaniese werkers bevestig op 'n interessante wyse die waarnemings wat aanvanklik op hierdie gebied deur Malkiel-Shapiro, Bersohn en Terner[1] gedoen is. Trouens, die Tasmaniese resultate dui op 'n selfs opvallender effek as wat oorspronklik deur die plaaslike navorsingswerkers gerapporteer is.

Die Suid-Afrikaanse waarnemers het nie alleen die moontlike kliniese waarde van magnesiumsulfaat by die behandeling van koronêre slagaarsiekte aangedui nie, maar het ook gewys op sekere biochemiese effekte wat deur hierdie behandeling teweeggebring word.

Dit is interessant om daarop te let dat Perlia[2] ook gewys het op die definitiewe kliniese waarde van parenterale magnesiumsulfaat by die behandeling van angina pectoris en koronêre trombose.

Na 'n sorgvuldige evaluasie van 50 gevalle van angina pectoris wat op hierdie manier behandel is, het Agranat[3] tot die slotsom geraak dat die gebruik van magnesiumsulfaat in gevalle van angina pectoris, op grond van die tans beskikbare inligting, ruimskoots die moeite werd skyn te wees.

Sandler en McGregor[4] wat 16 gevalle bestudeer het en, by die keuse van hul kliniese materiaal, gebruik gemaak het van maatstawwe wat verskil het van dié wat deur Malkiel-Shapiro, Bersohn en Terner toegepas is, het tot die gevolgtrekking geraak dat die verbetering wat waargeneem is ná die toediening van magnesiumsulfaat niks groter was as dié wat met 'n placebo bewerkstellig is nie. Malkiel-Shapiro en Bersohn[5] het hierdie aanspraak baie volledig en krities ontleed.

Bersohn[6] het 'n moontlike verduideliking van die waargenome effekte verstrek en aan die hand gedoen dat in magnesiummetabolisme ons bes moontlik die skakel kan vind tussen die aterogeniese en die trombogeniese bestanddele wat by intravaskulêre stolling betrokke is.

Anstall *et al.*[7] het onlangs gerapporteer dat magnesium, selfs wanneer dit mondeling toegedien word, 'n neiging toon om die koagulasie van die bloed te verleng, d.w.s. dat magnesium, uit die standpunt van die pasiënt, 'n definitief heilsame effek op die stollingsmeganisme van heelbloed het. In hierdie uitgawe bevestig Parsons, Butler en Sellars nie alleen die reeds beskrewe biochemiese veranderings in die lipiede patroon nie, maar toon ook aan dat magnesium die fibrinolitiese aktiwiteit van die bloed verhoog. Hierdie waarnemings vorm 'n skakel tussen die trombogeniese en die aterogeniese meganismes waarna reeds verwys is.

Daar moet beklemtoon word dat geen doeltreffend gekontroleerde kliniese studies i.v.m. hierdie terapeutiese aanspraak tot dusver uitgevoer is nie. Die Tasmaniese gegewens is die naaste wat daar tot sover gekom is aan 'n uitgebreide opname, want die waarnemings is oor 'n tydperk van 2 agtereenvolgende jare in dieselfde hospitaal gedoen. Gedurende die eerste jaar was die sterftesyfer in die geval van pasiënte wat aan koronêre slagaarsiekte gely het en met 'n anti-stollingsmiddel behandel is, 30%. Gedurende die volgende jaar, toe parenterale magnesiumsulfaat bygevoeg is, was die sterftesyfer 1%. Dit, tussen hakies, bevestig die oorspronklike Suid-Afrikaanse aansprake in verband met die kliniese effekte.

Hoewel die kliniese saak nog nie definitief bewys is nie, skyn dit asof daar 'n steeds toenemende massa getuienis is, opgelewer deur proefnemings met diere sowel as met die mens, wat daarop dui dat magnesiumsulfaat bes moontlik 'n belangrike terapeutiese effek op hierdie besonder gebied kan hê.

Dit is beslis wenslik dat kliniese proefnemings op 'n uitgebreide skaal en onder behoorlik gekontroleerde toestande uitgevoer moet word om hierdie interessante veronderstelling te toets.

1. Malkiel-Shapiro, B., Bersohn, I. en Terner, P. (1956): Med. Bydr., **2**, 455.
2. Perlia, A. H. (1956): Sovetsk. Med., **20**, 63.
3. Agranat, A. L. (1958): Med. Bydr., **4**, 67.
4. Sandler, A. en McGregor, M. (1958): S. Af. Tyd. G., **32**, 697.
5. Malkiel-Shapiro, B. en Bersohn, I. (1958): S. Af. Tyd. G., **32**, 1031.
6. Bersohn, I. (1958): Med. Bydr., **4**, 62.
7. Anstall, H. B., Huntsman, R. G., Weitzman, D., Lehman, H. en Hayward, G. H. (1959): Lancet, **1**, 814.
8. Parsons, R. S., Butler, T. en Sellars, E. P., (1959): Med. Bydraes, **5**, 487.

CHLORPROPAMIDE IN THE NATAL INDIAN

A PRELIMINARY SURVEY

G. D. CAMPBELL, M.B., M.R.C.P. (EDIN.)
Durban

One of the newer anti-diabetic sulphonylureas is chlorpropamide (1-p-chlorbenzene-sulphonyl)-3-propylurea). Chemically it is similar to the earlier sulphonylureas, differing only in having a shorter side chain, and a chlorine atom in the para position. It is a stronger hypoglycaemic agent that tolbutamide (having a half-life of 35 as against 3.5 hours).[1] In doses of over 500 mg. daily, a number of side effects have been described. Tablet for tablet, it is more expensive than tolbutamide, 3 tablets of tolbutamide costing the same as 2 of chlorpropamide; but chlorpropamide is generally prescribed in a lower maintenance dose due to its more powerful action. The Natal Indian is particularly 'resistant' to all anti-diabetic measures for various reasons, and the present trial of chlorpropamide was started 10 months ago to see whether it would be as good as, or better than tolbutamide and metahexamide in the Natal Indian diabetics, on whom trials had been run concurrently. Of a total series of 420 patients at present on trial with chlorpropamide, 207 have been followed for between 5 and 10 months, and form the present study.

THE NATAL INDIAN DIABETIC

Though it is impossible to assess accurately the incidence of diabetics in Natal Indians, there is probably no racial group so liable to the disease. As a measure of the commonness of their diabetes, Cosnett[2] has said, in the course of a study of a large number of admissions to the Indian Medical Wards, that 'diabetes is the single commonest medical cause for admission to the medical unit.' Without any effort, there have been collected into the Diabetic Clinic of the King Edward Hospital no less than 1,200 diabetic patients in the 14 months of the Clinic's existence: of these 950 have been Natal Indians. We still sign on 15 to 20 new patients every week, and this flow shows no signs of abating.

The Natal Indian's diabetes is characterized by two main features—the clinical mildness of his condition, and his resistance to treatment. This mildness is borne out by the fact that diabetic coma, even in patients who are 'insulin-dependent' who stop taking insulin, is almost unknown. In spite of this apparently mild diabetic state, the Natal Indian develops, with dramatic speed in some cases, the most severe vascular complications and it is not uncommon to see patients presenting for the first time for treatment of their condition with a fully developed nephropathy of the Kimmelstiel-Wilson type, with albuminuria, hypertension and marked retinopathy. Recently, a patient presented with dimness of vision due to an advanced proliferative retinitis, having never been conscious of being a diabetic. We see, quite often, a diabetic retinopathy in non-diabetic patients—whether these are in fact pre-diabetics only time will show. For full clinical details of 207 patients, the reader is referred to Cosnett's excellent paper.[3]

In the Natal Indian there is a very wide spectrum of economic consequence. It is well known that there is a great number of affluent Indians in Durban. In contrast to these, enormous numbers are distressingly poor and illiterate, and it is for obvious reasons that we see so many of these latter people in the King Edward Diabetic Clinic, as the Hospitals cater mainly for those of the poorest strata. Surprisingly few of our patients can speak English, and the direct doctor-patient relationship (which is so important in dealing with underprivileged diabetics) can only be established with the use of 'Fanagalo' Zulu, which is spoken by almost every Natal Indian. In contradistinction to the Zulu diabetic,[4] where diabetes is seen chiefly in patients from the upper and middle social levels, the disease in the Natal Indian is as common in the shack dweller as it is in those who live in mansions. Consequently, our Natal Indian diabetics are difficult patients, most of them being illiterate, dietarily inflexible (or too poor to buy non-carbohydrate foods anyway), completely without insight into the potential seriousness of their condition, and coming from homes in which sterile syringe technique would be a complete impossibility. For obvious reasons, the oral anti-diabetic agents have been a great boon.[4-6] Virtually the only dietary changes that we have been able to impose on most

of our patients, is to substitute saccharine liberally for sugar, to which they are so partial. It is obvious that in this rather patriarchal racial group, one of the few outlets that any of the home-cooped women have is that of excessive eating, particularly of filling carbohydrate foods, and they are very jealous of this pleasure!

Insulin resistance occurs at all ages in the Natal Indian.[3] In addition, control is difficult in people who will stop taking insulin if their urines are free of sugar, or who take insulin once weekly, because they are 'too poor.' Trials with the oral anti-diabetic agents were started in the Natal Indian with great hopes that they would be effective and, to that end, was converted as many patients as was possible from insulin to oral therapy.[5] To date we have had over 350 Indian patients on tolbutamide, and have encountered the following side effects: mild hypoglycaemia, nausea, troublesome headaches and skin rashes. One disadvantage of tolbutamide in the Natal Indian, is the commonness of 'resistance' to the drug, and the high maintenance dosage (3,000 mg. daily) that many patients require; 86 Natal Indians were followed for 5 months on metahexamide, which proved to be an effective hypoglycaemic agent. The trial, however, was stopped because of frequent side effects.[11]

In this trial it was intended to see if chlorpropamide would have fewer 'resistances,' and whether maintainance cost per patient per day would compare more favourably with that of other oral agents. In particular, in view of reports of side effects in other series, special interest was taken to assess the possible toxic effects that might be seen with chlorpropamide in the Natal Indian.

TYPES OF DIABETICS IN THE SERIES

In a clinic that has several types of diabetics, the use of the terms 'Type One' and 'Type Two' is avoided for obvious reasons. The types seen in the Clinic comprise the following, and they are defined as follows:[4-6]

Senile. Onset generally over 60 years; mostly thin, and satisfactorily controlled on oral therapy, or a small dose of insulin. Drug treatment necessary, however well the patient 'diets.'

Middle-Aged. Age of onset generally over 40 years; mostly fat, and dietary control almost impossible, though many are satisfactorily controlled on oral therapy. These form the bulk of the present series.

'J' Type Diabetics.[3, 7, 8] Generally under 40 years at onset; mostly thin and requiring more than 80 units of insulin daily. Not liable to ketosis if insulin is withdrawn, but often become unwell. Drug treatment is desirable. Seen in the Indians only, and not in Africans, as in Jamaica.[7]

Young Insulin-independent Diabetics. Generally under 35 years at onset, 50% being overweight. Satisfactory response to oral therapy, if started early.[8] Many of these may come into the category of 'J' types. To make the diagnosis of 'J', it is essential that the patient's insulin requirements be established. Because so many of these young patients, fat or thin, have responded so well to oral therapy, insulin requirements have not been established. Being overweight, however, rules patients out of the 'J' type.

Young Insulin-Dependent Diabetics. (In Europeans, sometimes referred to as 'Type One.') This is the classical young thin insulin-sensitive diabetic, who is labile, and goes into ketosis or coma if insulin is withdrawn. Very uncommon in the Natal Indian.

Chronic Pancreatitis Patients. No pancreatitis patient was included in the trial.

(At present, 6 established diabetics who are pregnant, are on chlorpropamide, of whom two (1 failure, 1 success) are included in the trial):

TABLE 1: SHOWING TYPES OF DIABETICS IN 207 NATAL INDIAN DIABETICS ON CHLORPROPAMIDE

	Males	*Females*
Senile	7	19
Middle Aged	40	107
True "J" Type ..	2	1
Young Insulin-Independent	4 } =6	26 } =27
Young Insulin-Dependent	–	1

Table 1 shows that most patients are middle-aged females.

METHODS OF TREATMENT AND CRITERIA OF CONTROL

It must be emphasized that this is a purely clinical study. Lack of facilities, grossly inadequate staff and weight of numbers of patients have precluded completely the blood sugar stabilization of our patients. As many as can, are encouraged to test their urines once

or twice daily: illiterate patients are asked to bring relations that can read or write, who learn to test urine, and are given the necessary equipment to do so; *pro formas* to record the results, are given as well. The Natal Indian patient, in sharp contradistinction to the Zulu diabetic, is a notoriously bad witness about his condition, as the extent of any symptom is grossly overestimated. It is an excellent rule to say that if they have no complaints and are satisfied with therapy, then they may be regarded as having improved.

Assessment of control in series of co-operative patients depends very much on the degree of dietary restriction in each patient. In most Indian diabetics this assessment is easy to make, any dietary restriction being negligible. Consequently in the present series, two criteria of control are used: 'Satisfactory' and 'unsatisfactory.' Into the criterion of 'satisfactory' control come patients who have been followed for over 5 months on chlorpropamide; whose subjective improvement has been such that the patient is satisfied with the treatment; in whose urine there has been a disappearance or significant decrease of sugar, and who have not developed side effects of the order necessitating stoppage of the drug. This group of 'satisfactory control' has been broken down into 'good' and 'fair.' Into the second category of 'unsatisfactory,' come patients who have had to be taken off the drug.

DRUG DOSAGE AND ADMINISTRATION

In view of the fact that many reports[9] have shown that side effects were seen more frequently in patients who were on doses of chlorpropamide of more than 500 mg. daily, it was decided to restrict dosage to this level in all patients. All those above the weight of 120 lb. were started on an initial dose of 500 mg. daily, and those below this weight on 250 mg. The dose was given in 2 divided daily doses—as in this group of patients, twice daily dosage is always psychologically more effective than a single dose, though a single daily dose has been postulated as being sufficient with chlorpropamide, as regards its hypoglycaemic action and its level in the serum.[1] Some patients took the drug in excess of the stated dose through not having understood instructions. One took 1,500 mg. daily for 10 days without ill effects. The only case which developed a skin rash had taken 1,000 mg. daily for 2 weeks (see below). Maintenance dosage in all cases requiring the drug lay between 250 and 500 mg. daily.

In the present series of Natal Indian diabetics, the following degrees of control were obtained (Table 2). The criterion of 'satisfactory' control, is subdivided into 'good' and 'fair': 'unsatisfactory' indicates those patients who had to be taken off the drug for resistance or side-effects.

TABLE 2: RESULTS OF TREATMENT WITH CHLORPROPAMIDE IN 207 NATAL INDIANS

	Males		*Females*	
Good	31	50	105	152
Satisfactory				
Fair	19		47	
Resistance	1	1	3	4
Unsatisfactory				
Side Effects	–		1 (Dermatitis)	

Satisfactory control was achieved in 202 of 207 patients who had been on chlorpropamide for 5—10 months.

The maintenance dose in 49 patients was 250 mg. daily, and in 153 patients 500 mg. daily.

TOLBUTAMIDE-RESISTANT PATIENTS

In the present series of 207 patients who had been satisfactorily followed for between 5 and 10 months, 24 had been resistant to tolbutamide, and 5 had been taken off that drug because of side effects. Previously, tolbutamide resistance was regarded as being poor control on a dose of 2 tablets *t.d.s.* (3,000 mg. daily). After following our tolbutamide patients further, it could be seen in the Natal Indian that if the patient was resistant to 1½ tablets *t.d.s.* (2,250 mg. daily), then he was generally resistant to the larger dose. Of the 24 resistant patients, 13 had been on 3,000 mg. and 11 on 2,250 mg. daily. On converting these patients to chlorpropamide, all were well controlled: 14 on 500 mg. daily, and 10 on 250 mg. daily. The side effects for which patients were changed from tolbutamide to chlorpropamide were: severe headache (3 cases); skin rash (1 case); and generalized pruritus (1 case). These side effects disappeared on changing to chlorpropamide, and all 5 patients were controlled on 250 mg. daily.

METAHEXAMIDE-RESISTANT PATIENTS

Patients who were not satisfactorily controlled on 150 mg. (1 tablet *t.d.s.*) of metahexamide daily, were regarded as being resistant to the drug. Seven patients came into this group and were changed to chlorpropamide; 4 were

controlled on 500 mg. and 3 on 250 mg. daily. Three other patients who developed side effects due to metahexamide (one with a rash, and 2 with hypoglycaemia, one being severely hypoglycaemic) were changed to chlorpropamide, 2 being controlled on 500 mg., and one on 250 mg. daily—the side effects disappearing satisfactorily.

CHLORPROPAMIDE FAILURES

In the series of 207 patients, 5 had to be taken off the drug for various reasons. One was a 23 year 'J'-type female patient who responded neither to tolbutamide or chlorpropamide, and had to be put back on to big doses of insulin. Partial failure was seen in a thin 17-year 'J'-type male who, after having 240 units of insulin for 3 years, had 3 months satisfactory control on 500 mg. chlorpropamide daily. At the end of this time, he began to lose weight and to pass large amounts of sugar in his urine; since he has been controlled on 500 mg. daily, with 60 units of lente insulin. A thin 70-year-old senile diabetic, proved to be better controlled on 1,500 mg. tolbutamide daily, than on a daily dose of 500 mg. of chlorpropamide. Attempts to control a 32-year pregnant diabetic who was difficult to control on insulin, with 500 mg. daily, was a complete failure though at the time of writing 5 other pregnant young women (one included in the series, and 4 who are not) are satisfactorily controlled. A fat 40-year female with diabetic gangrene became resistant to the drug after an initial period of fair control of 3 months, coincident with improvement in her gangrene. One patient was taken off the drug because of a skin rash (see below).

SIDE EFFECTS

In the Natal Indian, the following side effects have been encountered with the sulphonylurea drugs. With tolbutamide (360 patients followed 6–14 months): nausea and headaches, and skin rashes;[10] and mild hypoglycaemic episodes.[8] With metahexamide (86 patients followed for 5 months)—skin rashes and severe exfoliative dermatitis, mild and severe hypoglycaemic episodes, and a type of 'burning foot' syndrome.[11]

In the chlorpropamide symposium[9] of the New York Academy of Sciences (1958), toxic reactions were described as 'common' in doses exceeding 500 mg. daily. There appeared to be little point in going beyond this dose as, if patients are going to respond, they will do so to 500 mg. Among the side effects described are mild gastro-intestinal effects,[12] skin reactions and hypoglycaemia[13] and cases of obstructive jaundice.[14] In a recent paper, Granville-Grossman *et al.* summarized by saying: 'Chlorpropamide has been used widely, and no serious toxic effects have been so far reported.'[15]

The following side effects were encountered in the present series:

Skin Rash: A single case of classical sulphonylurea dermatitis was seen in a 48-year female patient, who took in error 1,000 mg. daily (twice the prescribed dose), because she misunderstood instructions. This faded, with withdrawal of the drug, but recurred when the drug was restarted in a dose of 250 mg. daily. A remarkably similar but severe rash was seen in a patient on 500 mg. daily, who, having misunderstood her general practitioner's instructions, swallowed several arsenical vaginal pessaries. This rash faded on treatment, in spite of the fact that chlorpropamide was not discontinued.

Hypoglycaemia. Two patients developed mild hypoglycaemic episodes on 500 mg. daily, which disappeared when the dose was lowered to 250 mg. daily, control still being satisfactory.

Abdominal discomfort and nausea. Two patients ascribed these symptoms to the drug, but on being persuaded to carry on on the same dosage, these symptoms disappeared. No cases developed 'sulphonylurea headache,' burning feet or jaundice.

SUMMARY AND CONCLUSIONS

In a preliminary study of the effects of chlorpropamide on the diabetic control of 207 Natal Indian diabetics followed from 5 to 10 months, most encouraging results were obtained. Of these patients, only 5 had to be taken off the drug, and of these 5, only 2 would come into the category of 'properly-selected' patients, i.e. suitable middle-aged or senile diabetics.

The difficulties in dieting and educating penurious and illiterate diabetics are discussed and the natural advantages of the use of the oral drugs in these patients stressed. One of the criteria of control is that the patient should be satisfied with his treatment. This series of patients is taken from a total of 226 patients, of whom only 19 (less than 10%) defaulted on any occasion over the 5–10 months' trial.

It would appear that chlorpropamide is a most effective hypoglycaemic agent in the Natal Indian, particularly in respect of patients

resistant to tolbutamide or metahexamide. It is an entirely safe drug, side effects under the daily dose of 500 mg. being negligible. Maintainance dose cost (25% of patients on 250 mg., and 75% on 500 mg. daily) of chlorpropamide would appear to compare favourably in this series with that of tolbutamide (70% of patients being on 1,500–2,250 mg. daily); 500 mg. of chlorpropamide being equivalent in cost to 1,500 mg. of tolbutamide.

Grateful thanks go to Dr. S. Disler, but for whose help the Diabetic Clinic would not exist. I would like to thank him for permission to publish details about these cases. Dr. W. G. McNeill of Eshowe was intimately concerned with the earlier months of the trial and I am very grateful to him for his help and encouragement; in the latter months Dr. James Davidson has taken over work on the trial and I would like to thank him. I am anxious to acknowledge the great kindness of the many Consultants of the King Edward Hospital in referring their diabetics to the Clinic, in particular Dr. N. Lamont, who kindly checked the manuscript. I would like to give special thanks to Sister Lowe, in charge of the Medical Out-Patients Department and, in addition, to Staff Nurses Tabezile and Beauty.

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SPOROTRICHOSIS TREATED WITH GRISEOFULVIN

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Griseofulvin is now accepted as an efficient systemic drug in the treatment of superficial dermatomycoses. The brochures of both British manufacturers* stress the fact that this antifungal antibiotic owes its successful action to its deposition in epidermal keratin. It is thus brought into contact with dermatophytes living in hair, nails and the horny layer of the skin. Both firms also state that its action is confined to the dermatophytes and that it is unlikely to be of value in the treatment of deep fungous infections (blastomycosis, histoplasmosis, actinomycosis, cryptococcocis, coccidioidomycosis, madduromycosis) or moniliasis. This conclusion was supported by Blank and Roth,[1] who reported that infections caused by *Candida albicans, Malassezia furfur,* and blastomycosis dermatitidis did not seem to respond to this therapy.

Recently this assumption has been questioned by Latapí and his co-workers.[2] With the oral administration of griseofulvin they obtained clinical cures of a case of mycetoma due to *Nocardia brasiliensis* in 2 months and another of sporotrichosis due to *Sporotrichum schenkii* in 4 weeks. This report prompted the writer to use griseofulvin in the treatment of sporotrichosis in the Transvaal.

*Griseofulvin is marketed in South Africa as Fulcin (I.C.I.) and Grisovin (Glaxo Laboratories). Both firms supplied the writer with generous quantities of their product.

CASE REPORTS

1. A White male aged 48 was seen on 3 September 1959 with a sore on the left hand of 2 months' duration. He did not remember a previous injury, but his job as bricklayer exposed his hands to minor trauma.

There was a plaque-like granuloma on the back of the left hand and 5 bean-sized nodules were clearly visible on the left forearm in the line of lymphatic drainage. A fragment of tissue removed from the primary lesion produced a growth of *Sporotrichum schenkii* in 15 days. Treatment with griseofulvin, 250 mg. by mouth 4 times daily, was begun on 3 September. Two weeks later clinical improvement was noted and on 15 October neither the granuloma nor the nodes could be palpated, though some bluish-red staining could

be seen. After a further 2 weeks on the same dosage the hand and the arm appeared completely normal and administration of the drug was stopped. There has been no recurrence in 4 weeks.

2. On 15 September 1959 a splinter from a pit prop lacerated the right wrist of a White miner aged 44 years. About 2 weeks later a nodule appeared on the healing wound and on 13 October 1959 he presented a chancriform ulcer about 2 cm. in diameter. No nodules were felt in the area of lymphatic drainage. Culture of pus produced a growth of *Sp. schenkii.*

Griseofulvin, 250 mg. 4 times daily, was given from 13 October. The ulcer began to heal within a few days and on 3 November was completely epithelialized, with the exception of a dry central scab measuring 3 mm. in diameter. Griseofulvin was continued for another 2 weeks, after which a healthy, pliable scar was the only visible departure from normality. The drug was then discontinued. Sufficient time has not yet passed to exclude the possibility of a relapse.

COMMENT

The case of Latapí *et al.* and the 2 cases presented here suggest that griseofulvin may prove to be the treatment of choice in sporotrichosis. The only previous efficacious treatment, with large doses of potassium iodide, usually requires from 2 to 3 months to effect a cure, and is extremely unpleasant to most patients.

SUMMARY

Two cases of sporotrichosis were treated with oral griseofulvin with satisfactory results.

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HYPOTHERMIA

A REVIEW

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It is proposed to discuss the physiology of hypothermia and its clinical application, mentioning the hazards associated with this technique. Methods of producing hypothermia will only be mentioned, without discussing details of these procedures.

Definition: Hypothermia means a fall in body temperature below its normal resting level. Strictly speaking, therefore, any temperature below 37°C. should be regarded as hypothermia, but the term is usually restricted to temperatures of 34°C. or lower.[1]

METHODS OF PRODUCING HYPOTHERMIA

1. Body surface cooling.
2. Pervascular or Extracorporeal cooling.
3. Body cavity cooling.
4. Selective cooling of organs.

Churchill-Davidson states that it is not the method of cooling used, but the physiological response to hypothermia which is of vital importance.[1]

PHYSIOLOGY OF HYPOTHERMIA: METABOLISM

With a fall in body temperature, there is a reduction in metabolism, and therefore in oxygen uptake by the cells. It has not yet been decided whether this fall in oxygen consumption is a linear[2] or exponential[3] function of the body temperature. This problem will be solved when it is possible to measure an accurate 'mean' body temperature. At present oxygen consumption is calculated against a temperature recorded at a single so-called representative body site.

There is little evidence to show that tissue oxidative enzymes are inactivated by surgical hypothermia.[4] It is important to realize that oxygen will be less readily available to the tissues, because of a shift to the left of the dissociation curve of oxyhaemoglobin at low temperatures.

This increase in resistance to oxygen lack seen in hypothermia is not the same for all

tissues.[5] The brain becomes more resistant to anoxia than the heart when it is cooled, whereas at normal temperatures the heart will stand more anoxia than the brain. This is probably because of an oxygen debt incurred by the heart, which does not stop beating immediately the circulation is interrupted.

Different parts of the body cool at varying rates, depending upon position, blood supply and type of tissue. Rectal temperature recordings should therefore not be regarded as an index of the temperature in other body sites, as it may be up to 3°C. higher than the heart temperature during cooling.[5] Under these rapidly changing conditions, the oesophageal temperature at heart level is the most reliable index of the temperature of the heart and blood.[6]

The temperature of the brain during hypothermia cannot be recorded directly, except during neurosurgery. A thermometer in the pharynx gives an idea of the temperature of blood returning from the brain, and is usually 2 to 3°C. above that of the heart.[5]

How far the temperature should be reduced depends on the length of time it will be necessary to cut off the cerebral circulation. Evans and Gray state that at 28°C. 6 minutes of circulatory arrest can be allowed, with 10 minutes as the absolute maximum.[7]

It is, however, increased myocardial irritability at low temperatures which limits the application of this technique.

DEEP HYPOTHERMIA

If it were possible to control the dangers of hypothermia, the period of circulatory arrest could be extended beyond 10 minutes.

A team at Westminster by separately cooling the blood flowing to the left and right sides of the heart and returning it through cannulae in the pulmonary and femoral arteries, relieved most of the strain on the heart during cooling. Using this technique, temperatures around 15°C. were achieved in human patients. This allowed circulatory occlusion for at least 45 minutes.[8]

The merits of this technique await future evaluation.

EFFECT OF HYPOTHERMIA ON THE CARDIO-VASCULAR SYSTEM

Bradycardia is a characteristic feature of cooling, and is probably due to a direct effect of cold on the sino-atrial node.[9] Depression of conduction in the bundle of His will prolong systole. The following E.C.G. changes occur:

i. Prolonged P-R interval.
ii. Spreading of the QRS complex.
iii. Elevation of the S-T segment.
iv. Auricular fibrillation is not infrequently seen during hypothermia.[7]

Coronary blood flow is greatly reduced during hypothermia.[10] Nevertheless it has been shown that the oxygen supply to the myocardium is adequate, provided the heart is beating normally. CO_2 elimination also presented no difficulty.[11, 12]

The Cardiac Output falls as body temperature is reduced. Vasoconstriction will maintain systolic blood pressure in the early stages, and so ensure adequate coronary blood flow, but at ± 28°C. this compensatory mechanism fails, resulting in hypotension with an ineffective coronary circulation. The use of vasopressors such as noradrenaline has been suggested, but these are difficult to control.[13]

The work done by the heart per minute is reduced, by cooling, to about one tenth of normal, the work per stroke is only reduced by one half.[4]

Ventricular Fibrillation is the most dreaded complication which can arise during hypothermia. The following factors will influence its onset:

(*a*) *Mechanical irritation* by surgical manipulation or catheters in the ventricle.[14]

(*b*) *Respiratory acidosis* due to underventilation will predispose the heart to ventricular fibrillation.

Especially dangerous are sudden changes in pH, such as may be found when the clamps used for occluding the venae cavae during surgery are removed. Brewin *et al.* claim that this circulatory occlusion during hypothermia results in marked metabolic acidoses due to lactacidaemia.[15] Swan *et al.* advocated hyperventilation which, in producing respiratory alkalosis, probably counteracted this metabolic acidosis and afforded protection against ventricular fibrillation.[16]

(*c*) *Poor coronary filling,* which may follow on an abrupt fall in systemic blood pressure, can precipitate ventricular fibrillation because of myocardial hypoxia. The shift to the left of the oxyhaemoglobin dissociation curve during hypothermia can possibly further aggravate tissue hypoxia. Hypocapnia due to deliberate hyperventilation can, for the same reason, further impede release of oxygen to the tissues.

(*d*) The relative concentrations of calcium and potassium ions in the blood are also of importance in the onset of cardiac arrhythmias.[17]

(*e*) Deep anaesthesia also precipitates ventricular fibrillation. Of the thiobarbiturates, thiopentone is the least likely to cause fibrillation.[18]

Numerous drugs and techniques have been suggested to prevent ventricular fibrillation during hypothermia, e.g. prostigmine,[19] procaine amide,[20] infiltration of the sinoatrial node with procaine,[21] depriving the heart of its sympathetic nerves,[22] all with disappointing results in clinical practice.

VASOCONSTRICTION AND SHIVERING DURING HYPOTHERMIA

Vasoconstriction constitutes the body's delicate and primary reaction to cold. Burton *et al.* state that this is due to a direct action of cold on the blood vessels as well as reflex in origin.[23] Samson Wright holds the view that vasoconstriction is caused by adrenaline released reflexly in response to the cold stimulus.[24]

If vasoconstriction is not controlled it will delay cooling, because blood will be diverted from the cold skin. The main blood stream is thus prevented from coming in contact with the cooling agent, and a steep temperature gradient is set up between surface tissues and the body core.

Two undesirable effects then arise:

i. So-called 'after-cooling,' because warm blood from the interior will continue to flow through the cold surface tissues when the cooling agent is removed. The temperature of internal organs will consequently continue to fall.

ii. During re-warming the sudden increase in the capacity of the vascular bed because of vasodilatation following on vasoconstriction, together with a reduction in plasma volume found during hypothermia might lead to peripheral circulatory failure.[25]

Shivering represents the coarse reaction of the body to a cold stimulus. Its intensity will be proportional to the size of the temperature gradient between the surface and the centre of the body. It is important to realize that a general muscular hypertonicity may precede visible shivering for a considerable time. This was shown by electromyographic studies, which revealed that muscle potential activity occurred soon after exposure to cold and increased in frequency until visible shivering could be observed.[26]

It is imperative that shivering and vasoconstriction should be obtunded if cooling is to be successful. Shivering increases metabolism and, consequently, oxygen consumption, thereby defeating the whole purpose of hypothermia.

Other undesirable effects include a rise in blood pressure and pulse rate. The rise of pulse rate at low body temperatures, in the absence of any other explanation, may be regarded as a guide to the imminent onset of shivering.[27]

Fortunately it has been shown clinically that the same drugs can be employed for the control of both vasoconstriction and shivering, e.g. light anaesthesia with muscle relaxants; chlorpromazine.

THE EFFECT OF HYPOTHERMIA ON THE CENTRAL NERVOUS SYSTEM

Rosomoff listed the following effects of hypothermia on the central nervous system:[28]

i. Decrease in cerebral blood flow at a rate of 6.7%/°C.

ii. Reduction on cerebral metabolism, so that at 25°C. metabolism was one third of the pre-cooling level.

iii. Reduction in the mean arterial pressure could be maintained for long periods without ill effects. This could be regarded as 'an induced compensated hypotension.'

iv. There was a reduction in brain volume of 4.1% at 25°C. and cerebrospinal fluid pressure was lowered. The contraction of the brain made its handling during surgery easier.

Electroencephalographic tracings show little change in cerebral activity until the temperature reaches 31–35°C., when some decrease in amplitude and frequency occurs. The electrical activity declines still further and at 20°C. there is no activity recordable. During re-warming no new rhythm appears and the changes of cooling appeared in the reverse order with a lag of 1–2°C.[29]

The response of the respiratory centre to inhalation of 6% CO_2 at temperatures between 25° and 27°C., and at normal temperature, showed very little difference in animal experiments done by Cranston *et al.*[30]

During hypothermia there is a lowered threshold for initiation of epileptiform seizures produced by electrical stimulation and metrazol, especially below 30°C.[31]

Afferent nerves show a higher threshold for stimulation, but the response elicited is more pronounced and prolonged and is poorly synchronized. Consequently the central response to afferent stimuli is augmented during hypothermia.[32]

There is also a tendency for afferent stimuli which would elicit simple monosynaptic responses at ordinary body temperature, to give rise to reflexes with polysynaptic components.[33]

It has been suggested that visceral stimulation during surgery in cooled animals may result in such polysynaptic discharges in the autonomic nerves to the heart, and that this could partly be countered by an adequate depth of anaesthesia.[4]

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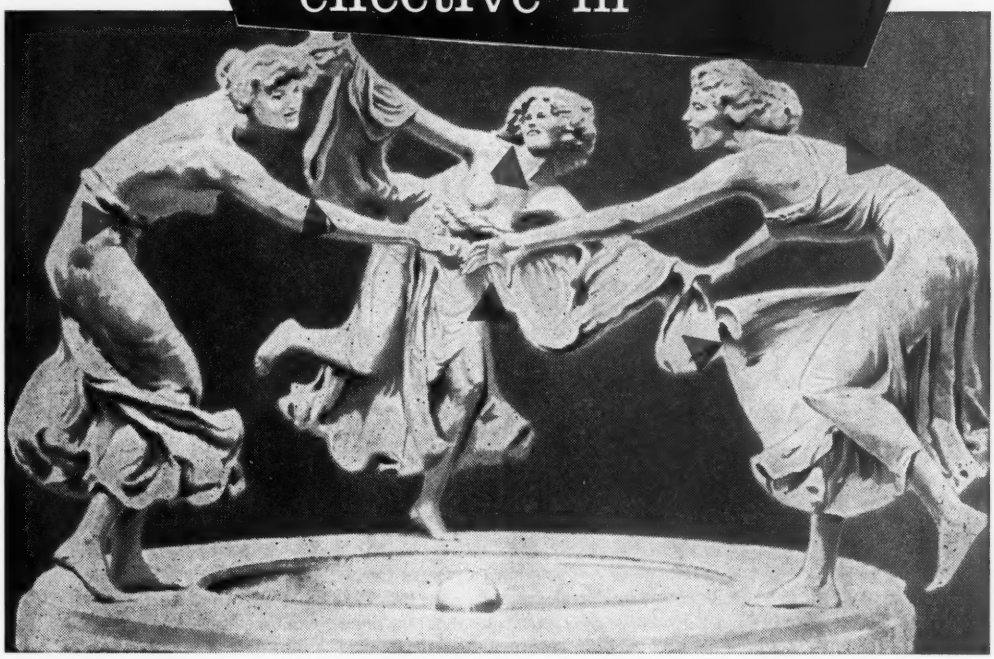

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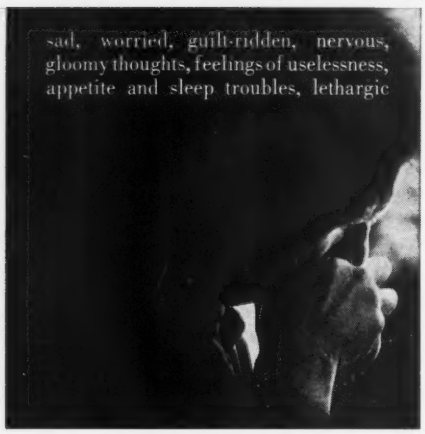

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THE RESPIRATORY SYSTEM

There is usually a tendency to develop acidosis when body temperature is lowered during hypothermia.[16] This was thought to be due to an increased solubility of CO_2 in the blood at low temperatures. Cranston *et al.* showed that this was not due to a failing respiratory centre.[30]

Brewin *et al.*, however, found evidence of a metabolic acidosis as well.[15] This was equivalent to a reduction of the CO_2-combining power by about 5 ml. per 100 ml. at a CO_2 partial pressure of 40 mm. Hg. This metabolic acidosis was aggravated by a period of 10-minute circulatory arrest. The blood lactate was also increased during hypothermia and a 10-minute period of circulatory arrest caused a further increase in the blood lactate level.

Swan *et al.* demonstrated that by hyperventilation the arterial pH could be kept up to normal levels, because alveolar and arterial CO_2 tensions were reduced by this manoeuvre.[16]

An increase in the physiological dead space during hypothermia, thought to be caused by bronchodilatation, has been demonstrated.[34] This did not produce trouble in CO_2 elimination.

LIVER FUNCTION DURING HYPOTHERMIA

During a short period of circulatory arrest in dogs under hypothermia, the central venous pressure rose considerably with consequent venous congestion in the liver.[15] This caused gross structural damage (centrilobular necrosis with extravasation of blood) and reduction of the galactose clearance test. The metabolic acidosis demonstrated by these workers was thought to be related to this structural damage in the liver. If central venous pressure was reduced during circulatory occlusion, liver structure remained normal, galactose clearance returned to normal and metabolic acidosis almost disappeared during re-warming. Hyperglycaemia followed by hypoglycaemia during hypothermia was thought to be due to exhaustion of liver glycogen stores.[35]

A reduced catabolism of aminoacids in the liver during hypothermia is probably responsible for a fall in blood urea.[36]

Changes that were found in liver function during hypothermia, were temporary and fully reversible.

RENAL FUNCTION AND CHANGES IN WATER AND ELECTROLYTE BALANCE

In induced hypothermia in dogs a decrease in glomerular filtration, tubular reabsorption, renal blood flow and urine volume was observed.[36] These investigators did not, however, find a consistent pattern of change.

It has been suggested that especially tubular secretion and reabsorption may be affected by the reduction in cellular activity during hypothermia.[37]

The kidney is protected to some extent by hypothermia against ischaemia brought about by complete occlusion of its blood supply.[38]

The residual damage which is present will depend on the duration of anoxia. Damage is prevented by a small, yet important blood supply via the lumbar collaterals.

Morales *et al.* found no evidence of permanent adverse effect on the kidney as a result of hypothermia.[39]

Extensive shift of fluids leading to increased haematocrit readings and blood viscosity, were observed in dogs cooled to 20°C.[40] Rodbard *et al.* demonstrated a reduction of little more than 30% in plasma volume in animal experiments. They concluded that blood volume was reduced by pooling in regions of low blood flow.[41]

Changes in plasma sodium and chloride are slight during hypothermia.[16, 35]

Plasma potassium levels were found to be raised by Bigelow *et al.*[42] and reduced by Swan *et al.*[16] and Nowill *et al.*[36] Wynn has pointed out the danger of a reduction in plasma electrolytes by dilution, if intravenous glucose is given during operations under hypothermia. This is caused by the impairment of glucose utilization and its removal from the extra cellular spaces, with a resultant increase of the plasma osmolarity.[43]

ENDOCRINE DISTURBANCES DURING HYPOTHERMIA AND STRESS REACTION

Endogenous production of insulin is reduced during hypothermia, and the ability of insulin to reduce the blood sugar is lowered.[35] The raised blood sugar levels following intravenous glucose infusion during hypothermia could not be lowered by administration of insulin.[43]

Thyroid activity is markedly reduced by body cooling, if shivering is prevented and an adequate level of anaesthesia is maintained.[44]

Pituitary ACTH secretion and antidiuretic hormone secretion are depressed during hypothermia.

Adrenal corticosteroid production has been reported to be reduced during hypothermia as well as adrenaline, noradrenaline and 17-hydroxycorticosterone secretion.[6] However, McPhee *et al.* found no marked reduction in corticosteroid production during hypothermia in dogs which lasted for 3 days.[45]

Re-warming restores these endocrine functions to normal, although it has been claimed by McPhee *et al.* that the production of 17-hydroxycorticosterone is higher on re-warming than in animals which are anaesthetized, but not cooled.[45] The severe changes found in the liver, kidneys and adrenals following hypothermia, induced by surface cooling, were thought to be due to a severe stress reaction.[46] However, using pervascular cooling, these changes were not found, and it was suggested that these tissue changes were not produced by hypothermia *per se.*

CHANGES IN THE BLOOD DURING HYPOTHERMIA

Blood pH. Lowering of the pH during hypothemia is thought to be due to an increased amount of CO_2 dissolved in the plasma[16] as well as a metabolic acidosis.[15]

Disturbances of the Blood Clotting Mechanism. Animals cooled by the surface method and kept at temperatures of about 29°C. for days, show a prolongation of the coagulation time, decrease of fibrinogen and reduced platelet counts, so that after 3 days the coagulation time may exceed half an hour.[65] It is postulated that some anti-coagulant substance is released.

Willson *et al.* found that in animals under hypothermia there was a decrease in procellerin, proconvertin, prothrombin, fibrinogen platelet thromboplastic factor and circulating platelets.[66] These changes all returned to normal or exceeded the normal level, during rewarming. Extensive studies in Man showed changes different from those found in dogs, a decrease in prothrombin composition being the most important change.[67]

Difficulty with clotting is particularly likely if blood is circulated through plastic tubes and the pump over a prolonged period with pervascular cooling. To avoid this danger the rate of cooling in adults should be 1°C. change of temperature every 5 minutes.[70]

During large transfusions with citrated blood which has been stored for a week or more, there is a serious risk of inactivating a large amount of the free calcium ions in the blood. This together with the increase of potassium ions and a lowered pH may lead to cardiac arrest. It has been shown that citrated blood stored for more than a week may have potassium levels as high as 8 mEq./L. and a pH of 6.4–6.8.[68, 69]

HAZARDS OF HYPOTHERMIA

While there are dangers associated with the hypothermic technique itself, it should be borne in mind that the hazards of any general anaesthetic still remain, e.g. the aspiration of stomach contents.

1. *Anoxic brain damage* during the period of circulatory arrest constitutes the chief hazard in cardiac surgery performed together with hypothermia. The temperature to which the patient may be cooled, together with the time permitted for circulatory arrest at that particular temperature, are the chief factors in the prevention of this complication. Ross states that evidence of brain damage was not found in cases cooled to 28°C. and in whom the period of circulatory arrest was less than 10 minutes.[47]

2. This hazard has been somewhat overshadowed by the *serious cardiac irregularities* found at low temperatures, ventricular fibrillation being the most important. It is this manifestation of increased myocardial irritability which has limited the full exploitation of the hypothermic technique. The aetiological factors have been discussed; most of them can be controlled by the anaesthetist, the surgeon or his assistants.[27]

In the repair of congenital cardiac anomalies care has to be taken with replacement of blood. A sudden increase in the work load of the ventricles, as may occur in the closure of an artrial septal defect, can precipitate cardiac failure. It is better to maintain a mild deficit in blood replacement.

3. *Coagulation Defects and Haemorrhage.* The retardation of coagulation during hypothermia has been stressed by certain workers and denied by others.[48, 49]

Dundee *et al.* reported intractable haemorrhage where pre-operative deficiency of the clotting mechanism existed in patients cooled by the surface method.[50] It is suggested that hypothermia will have more adverse effects in such patients than in patients with a normal blood clotting mechanism pre-operatively.

Another possible explanation of post-operative bleeding is the lowering of blood pressure occurring with cooling. Blood pressure tends to rise in the re-warming period and cause haemorrhage from previously dry vessels.[51]

The use of fresh blood for replacement therapy during surgery will tend to minimize the reduction in platelets.

Lewis *et al.* have remarked on the difficulty in diagnosis of hypovolaemic shock in the hypothermic patient, the normal response to a decreasing blood volume being masked by the effects of cooling on the cardiovascular system.[51]

This problem can probably only be solved by a rapid estimation of the blood volume e.g. by the use of radio-active cadmium.

4. *Frost-Bite and Other Skin Changes.* If extreme vascoconstriction during hypothermia is not countered, frost-bite may be caused.[7] Subcutaneous fat solidification and subcutaneous fat necrosis seen in infants cooled by the surface method, was thought to be due to difference in the chemical composition of fat in the infant.[51] There is a further risk of causing burns during active re-warming, following surface cooling.

5. *Hazards During the Re-Warming Period.* When surface cooling is used it may take up to 6 hours to re-warm a patient. This may be a disadvantage when rapid re-warming is required, especially if severe cardiac irregularity has occurred during surgery. Re-warming increased the chance of successful defibrillation.

The tendency of acidosis to develop during re-warming[52] and the role played by increased central venous pressure have been noted.[15] The prevention of a rise in venous pressure together with such measures as prevention of shivering during re-warming and the institution of spontaneous respiration as early as possible, will counter this tendency for the development of acidosis.[53]

6. *Delay in Detoxification and Excretion of Drugs.* Care must be taken to use minimal doses of drugs during hypothermia. Detoxification of morphine and barbiturates is slowed down considerably.

There is a potential danger that noradrenaline used during cooling may, on re-warming, cause undesirable hypertension. A similar risk exists as far as recurarization is concerned; the drug having been inactive in part in the circulation, becomes active during re-warming.[6]

INDICATIONS FOR HYPOTHERMIA

1. *Heart Surgery.* The limited time for circulatory arrest, together with the danger of ventricular fibrillation, limits the use of hypothermia to the repair of atrial septal defect and the relief of pulmonary stenosis in young subjects.[7]

The clinical usefulness of combined hypothermia and cardio-pulmonary bypass techniques in cardiac surgery remains to be determined.

2. *Surgery of the Great Vessels.* If the aorta has to be occluded above the origin of the renal arteries, hypothermia affords protection against anoxic damage to the spinal cord. Thus, Thornton has demonstrated that the duration of occlusion of the thoracic aorta can be extended safely from 20 to 60 minutes. He has also shown that hypothermia protects the kidneys during surgery of the aorta.[54]

3. *In partial hepatectomy,* where temporary occlusion of the hepatic blood supply is necessary, hypothermia will also afford protection.[55]

4. *Neurosurgery.* The removal of vascular tumours has been made safer by the combined use of controlled hypotension and hypothermia, the reduced cerebral oxygen consumption affording protection against adverse effects of induced hypotention.[56]

The safety factor afforded by hypothermia against the consequences of temporary or prolonged occlusion of major vessels, has made accurate repair of ruptured aneurysms, and arterio-venous anomalies possible.

If necessary, the period of hypothermia may be extended into the post-operative period for 12 to 24 hours. This temperature should not be lower than 28°C.[57]

5. Hypothermia has been employed in poor-risk cases undergoing extensive surgery and was found very necessary when hypotension was used as well.[58-60]

6. Therapeutic measures employing hypothermia include treatment of ischaemic brain injury, and hyperpyrexia following dysfunction of the hypothalamic heat regulating centre.[61]

Hypothermia may specifically prevent post-traumatic cerebral oedema, and protect the brain against ischaemia should oedema develop.[27]

Hypothermia has also been employed to counter hyperpyrexia found in association with acute polio-encephalitis and acute encephalitis.[62, 63]

In severe thyrotoxicosis hypothermia was used in pre-operative preparation and work done by Hunter suggests that hypothermia may be life-saving in post-operative thyroid crisis.[50, 64]

There is evidence that increasing use will be made of hypothermia as a therapeutic measure.[27]

OPSOMMING

'n Poging is aangewend om die fisiologie van hipotermie en die kliniese toepassing hiervan volledig te bespreek.

Die hoofgevare verbonde aan die gebruik van hierdie tegniek word breedvoerig uiteengesit en is kortliks as volg:

(*a*) *Ventrikulêre Fibrillasie.* Hierdie is die mees gevreesde komplikasie wat mag voorkom gedurende die toepassing van hierdie tegniek en mag veroorsaak word deur meganiese prikkeling van die hart, respiratoriese asedose, onvoldoende koronêre vulling, elektroliet wanbalans en diep narkose.

(*b*) *Bloedvaat Vernouing.* Hierdie komplikasie mag die verkoeling vertraag en wanneer die liggaam weer verhit word mag die vasodilitasie wat volg aanleiding gee tot perifêre sirkulatorieseinstorting.

(*c*) *Bewerasie.* Bewerasie verhoog die metalbolisme en dus ook die suurstof opname van die pasiënt en mag ook 'n verhoging in bloeddruk en polsspoed veroorsaak.

Hierdie komplikasie kan beheer word deur die gebruik van 'n ligte narkose met spierverslappers of deur die toediening van chlorpromasien.

(*d*) *Permanente breinskade* as gevolg van anoksie van die brein gedurende die periode van hartstilstand.

Tenslotte word die effek van hipotermie op die verskillende stelsels van die liggaam soos die sentrale senuweestelsel, asemhalingstelsel, ens., volledig uiteengesit.

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THE DOCTOR AS HEALER AND TEACHER

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'Doctor-oris *m.* (doceo) a teacher'. In Latin dictionaries will be found the root derivation and, one submits, the meaning, of our appellation. In addition to curing what is remediable and alleviating what is not, Medicine accepts the responsibility for preventing those diseases which need not occur. Are we as conscious and committed to the further goal of teaching the essentials of healthy living?

Most standard medical textbooks consist of excellent articles on diseases and their detailed management. Almost never do these details of management include the need for explaining to the patient the nature of the illness, the mechanism and significance of his symptoms and the course the illness may be expected to follow. This lack of attention to this aspect of management is often reflected in clinical teaching.

This paper will discuss the nature and sources of the health education to which the individual in our society is exposed and on which the doctor may impinge. The unique characteristics of general or family practice which favour health education and certain practice advantages of such educational procedures will be suggested. The objectives of the health education functions of the physician and the kinds of health problems which can be dealt with in family practice will be outlined and finally some of the barriers to the acceptance of this function will be noted.

SOURCES OF HEALTH EDUCATION

(*a*) *The Family.* A great proportion of the health behaviour of an individual is acquired within the family circle. Knowledge of, attitudes to, and action related to health and disease, are transmitted in this way from one generation to another. The mother is the first health educator of her children.[1] The family doctor caring for all members of the family can consciously and effectively become an important instrument in this transmission of health behaviour.

(*b*) *Public Health Practice.* Inherent in the preventive aims of public health practice is the need for health education. The traditionally accepted areas of public health concerned with maternal and child health, immunization, infectious diseases, sanitation and nutrition, are being extended to include chronic disease and mental health.

(*c*) *Industry.* The underlying motive of most of industrial health education is increased efficiency. It is therefore predominantly confined to workers and such health behaviour as is directly relevant to work situations. But as the importance of the family life situation and mental health of the worker becomes more evident so the educational scope of the industrial medical officer enlarges.

(*d*) *The School.* School health education should be regarded as a continuing influence running right through a school's daily life and

work.[2, 3] Of all the stages of human life, the time spent at school is the single, most important period from the point of view of systematic health education. Progressive school medical services pay much attention to this aspect of their work.[4]

(*e*) *Hospitals.* 'Especially in those countries where mothers are allowed to stay with their children and give them simple attention while they are patients, the hospital may serve as an important centre for health education activities.'[1]

For most individuals the impact of the hospital may be limited to short periods of acute disorder. There are nonetheless largely undeveloped areas of the health education functions of hospitals such as improved understanding and use of services including rehabilitative welfare agencies and changes in habits and way of life which will help to avoid recurrences.

(*f*) *Mass Propaganda Media.* It has been truly said that the source material for contemporary folk medicine and health education planning can be found in the mass propaganda media of Press, radio and television and screen.[5] Many of these run regular features dealing with medical and personal problems and interests and opinions. Properly used, these media can make some contribution to the general health education of a community. Their effectiveness, however, with the handling of the unique and particular problem of an individual case, is exceedingly problematic.

An interesting and promising development is the publication of the two journals *Family Doctor* and *Today's Health* under the aegis of the British and American Medical Asociations respectively. They maintain a very high standard of up-to-date and comprehensive health information presented accurately and simply.

THE DOCTOR, THE PATIENT AND HEALTH EDUCATION

Every medical act provides an opportunity for useful education.

'Treatment of illness is recognised as only a part of the total professional responsibility of the physician. His responsibilities are considered to include also those educational interventions which will contribute to the application of preventive measures by the individual and the families whom he serves. Experience has shown that physicians who respond to the concerns, anxieties, and curiosity of patients and their families find that a well-informed and educated patient is more likely to be a satisfied patient.'[1]

'The clinical situation, with its intimate doctor-patient relationship, is a peculiarly appropriate one for health education.'[6] This does not mean only 'a short didactic training in a limited mechanical procedure that has little meaning for them' and significantly called 'doctor's orders'. The advance of medicine makes it impossible to ignore the role of the total pattern of the individual's daily life in the etiology of disease and maintenance of health. 'The individual's personal influence on his own health is therefore of unusual importance.'

A common criticism of the physician of today is a seeming lack of sympathetic interest in his patients. There are those who in addition to their health tax under a National Health Service are prepared to pay a private practitioner's fees not on account of his greater technical efficiency but because they feel that they can expect more personal attention. The public may more or less take for granted the advantages of scientific diagnosis and treatment but in addition there is a definite desire for this personal attention.[7] Each patient wants to be regarded as an individual, not as an example of a particular syndrome. People come to their doctors because of concern about their symptoms. This anxiety is seldom completely allayed merely by the examination. To achieve this there must be time for listening to the story and explaining the illness and its general prognosis.

'The interest in what the doctor tells the patient has obscured the fact that communication is two-way, that what the patient tells the doctor may be just as important to the patient as what the doctor tells the patient. When the patient feels he can express his fears and preoccupations and receive both a sympathetic hearing, and guidance consistent with his values and life situation, diagnostic information becomes less important and less troublesome to both physician and patient.'[8]

One segment of public opinion (the opinion of the physicians themselves) provides a thought-provoking note. Curiously, physicians appear to have a worse opinion of themselves than their patients do. This was brought out in an American nationwide survey;[9] 21%, 46% and 80% respectively, of a group of 500 practising physicians thought that other doctors did not take enough personal interest in their patients, were not frank enough in talking to patients about their illnesses and do not give the patient as much time as the patient would like. From this survey there also emerged a picture of what people like about and expect from their doctors: sympathy, patience and understanding, rather than guaranteed cures and 'wonder drugs'.

The usually much neglected art of explanation is both a therapeutic tool and a professional obligation. 'The art of communication is an essential ingredient of the art of medi-

cine.'[8] In many diseases the care with which a patient carries out recommended treatment radically influences the efficacy of the therapy. Whether or not the patient cooperates to the desired degree depends to a great extent on his understanding of the need for the advised measures. Anxiety, which nearly every patient has, is a very formidable barrier to communication. 'It causes distortions, unwarranted shifts of emphasis, and inability to comprehend or remember or even to hear. If the physician wishes to be sure his patient understands what he is told, he must first allay disruptive anxiety.' He does this probably more by non-verbal than verbal means, by listening to the patient than by lecturing to him.

In some disorders even though the underlying source of tension may not yet have been identified or corrected, an explanation of the psychosomatic mechanism of the symptoms may materially assist improvement.

Each physician in his every contact with a patient strives to leave him the better for the encounter, but there are times when the patient is left the worse for the encounter with a harmful disorder unwittingly induced in him by the inappropriate use of drugs or faulty management or treatment *or because of the physician's examination, manner and discussion* or with, in two words, iatrogenic disease.'[10]

THE OBJECTIVES OF THE FAMILY DOCTOR'S HEALTH EDUCATION

The doctor needs to understand that just as his personality can be a cause in iatrogenic illness, so is it part of the cure. The doctor needs to develop and exploit a type of relationship with the patient 'which would include greater opportunities for self-expression by the patient and consequently a more passive and permissive role for the doctor, in which his interventions were not only explanatory, but suggestive and cooperative, rather than didactic and authoritarian.'[6]

The purpose of communicating information to the patient is not to make him a student of his own disease or to make him aware of all the threats to his health and survival, nor to arouse guilt and fear so that whatever results therapy achieves are vitiated by anxiety. 'Our purpose is really to achieve a state of mind in the patient so that he can cooperate fully in his treatment with a minimum of anxiety.'[8]

The patient can be given 'information to the extent that his motivation and understanding permits—which is somewhat further than most doctors are inclined to assume for most patients.'[6] If this greater degree of understanding is conceded to his patient, the doctor may also accept and exploit a similar greater degree of the patient's carrying responsibility for his own health care.

'The physician should encourage the patient to feel that he has control over his own destiny and that he has an intelligent role to play in his treatment and in the future care of his own health.'

'If a physician, or other medical person dealing with patients, assumes that all patients, regardless of education, intelligence or social and economic status, have the same values he has, he will find out only too frequently that he misunderstands and is misunderstood.'[8]

The physician must be alert to the frame of reference in which each patient operates, to avoid—'he talked over my head.'

Almost all patients carry as implicit but unformulated assumptions their own private science of physiology, which is usually erroneous and almost always incomplete, is a frequent source of anxiety and unnecessary invalidism and a potent barrier to effective communication.

'It is hard to convince a woman that she can resume her life activities if she believes that the removal of her breast has injured her heart and lungs. The physician must seek out and refute these assumptions, as they render information unacceptable, make the truth appear to be a lie and the doctor a liar. It must never be assumed that these assumptions do not exist or are not potent determinants of both communication and action.'[8]

The doctor can help to mould the expectations his patients have of him and the use they make of his services. They can have the security derived from experience that they may express their fears and anxieties without being considered foolish.

A general practitioner in the British National Health Service has reported on an interesting and promising experiment in health education in her family practice.[11]

'Mothers are taught the early signs of illness, when to send for the doctor, how to nurse a sick child at home, how to prepare a child for admission to hospital, what a child-guidance clinic is for, and much else which improves the mother-child-doctor relationship.'

She found that the rate of admission to hospital of children under 11 years went progressively down in 5 years from 2.6% to 0.4%.

THE SCOPE OF THE FAMILY DOCTOR'S HEALTH EDUCATION

The potential area in which the family doctor may exercise his health educational functions can extend to the limits of his enthusiasm and situational opportunities. Some examples of the range, variety and importance of the health

problems that can be dealt with will be briefly listed.

(*a*) *Childbearing, Childrearing and Family Planning.* This is the core function of the family and the family doctor can make a unique contribution to its healthy achievement. Particularly important is the role he can play in anticipatory guidance, namely discussing appropriate health behaviour well in advance, e.g. discussing childbearing and family planning with newly engaged or married couples, breastfeeding and the preparation of older sibs during pregnancy, and normal child growth and development during the early months of the infant's life. This allows time for the appropriate changes in attitude and behaviour to occur and thus prepare the individual for the tasks ahead. It also helps to prevent a repetition of previous inappropriate or undesirable health behaviour.

(*b*) *Nutrition and Dietary Habits.* Depending on the community within which he works various nutritional problems may present. Of particular importance to the family doctor are instances of food faddism. He has a very important role to play in combating the powerful influences of much publicised, and in many instances, poorly founded dietary regimes. He can do this by developing in his patients a healthy scepticism and discrimination.

(*c*) *Prevention of Disease and Accidents.* That the public want to learn of advances in preventive medicine is shown by its tremendous news value and rapid transmission. Here again the doctor can try to replace uncritical acceptance by enquiring initial disbelief. Where a new technique of prevention is validly proven it may be submitted that it is almost a moral obligation on every doctor to play his part in the transmission of this new knowledge to the public, e.g. for the prevention of poliomyelitis.

Home accidents, particularly child poisoning, could be much reduced if the family doctor becomes alert to the tendency in many families towards indiscriminate self-medication. Unless and until there is statutory control of medicament advertising, each practitioner will have to individually counteract the harmful effects of the present situation. The physician has a crucially important role to play in the reduction of road accidents. He must become alert to the effects of his patients' conditions and their treatments on their efficiency as drivers.[12]

(*d*) *Mental Health, Use of Leisure Time. Preparation for Old Age.* These aspects of living may be conveniently grouped together for the underlying principles on which the family doctor will base his health education are common to them. The front line defence position of the doctor in mental health has been dealt with in a previous paper.[13] The emphasis of medicine has moved from problems of dying to problems of living. The physician in addition to showing the people how to live longer must now also show them how to live together.

POSSIBLE OBJECTIONS AND DIFFICULTIES

Certain patients cannot question their doctors because of timidity, fear of appearing ignorant or apprehension about the answers. With experience an observant clinician can become intuitively aware of what the person under his care would like to know. Especially important is this in the numerous cases of 'cancer apprehensives'.[14]

The doctor may not be prepared to perform his educational function because of lack of interest[15] or lack of training.[16] Some resistance may be partly due to a belief that his status might be reduced if his pontifical omniscient and omnipotent manner were replaced by simple, colloquial, comprehensive and accurate explanation. Part of it may also be a hangover from the days when because of gaps in medical knowledge lack of precise information was unavoidable and the doctor tended to substitute for this an air of assurance and a skilful bedside manner.[7]

The ultimate solution of the problem, as of many others affecting modern medicine, probably lies in the field of basic medical education which may 'be designed to produce physicians who not only have the technical knowledge and skills of their craft, who not only see health and disease as a by-product of personal and social life, but who are at once humane and imaginative educators as well.'[6]

SUMMARY

The doctor impinges on many of the areas in which the individual in our society is exposed to health education.

Every medical act provides an opportunity for useful education.

The objectives and scope of the family doctor's health education functions are set out.

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NOTES AND NEWS : BERIGTE

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* * *

1959 FORMAN PRIZE

The 1959 Forman Prize has been awarded to Mr. D. G. Burns. Mr. Burns is Head of Smuts Hall, a member of the S.R.C. and President of the M.S.C. He has in no small way contributed to the smooth functioning of Students Clinics. It is in recognition of his fine contribution to such a wide variety of student interests that the award has been made to him.

1960 FORMAN AWARD

This has been made jointly to:

Dr. Neville Woolf, M.B., Ch.B. (Cape), M. Med. Path. (Cape), who is currently doing research in immuno-histochemistry at St. George's Medical School, London;

and

Dr. Marcus Jacobson, B.Sc., M.B., Ch.B. (Cape), who is currently doing research in neurophysiology at the University of Edinburgh.

* * *

THE NUTRITION SOCIETY

ADVANCE NOTICE OF 131ST MEETING

The following is the programme of a symposium to be held in London on 19 March, 1960 on *Nutrition and the Elderly.*

Chairman (*morning session*): Professor R. C. Garry (*Glasgow University*); (*afternoon session*): Professor R. E. Tunbridge, *O.B.E.* (*Leeds University*).

Professor K. J. Franklin, F.R.S. (formerly St. Bartholomew's Hospital Medical College): *The physiology of ageing.*

Dr. C. A. Boucher, *O.B.E.* (Ministry of Health, London): *The social medicine of old age.*

Mrs. D. W. Newman (Surrey Old People's Welfare Committee): *The social aspect with reference to nutrition.*

Dr. E. Woodford-Williams (General Hospital, Sunderland): *The clinical aspect of old age.*

Dr. Alex Comfort (University College, London): *Nutrition in relation to age changes in animals.*

Dr. B. E. C. Nordin (Gardiner Institute, Western Infirmary, Glasgow). *Osteoporosis and calcium deficiency.*

Dr. C. J. Earl (The National Hospital for Nervous Disease, London, W.C.1): *Nutritional disorders in the nervous system.*

Dr. J. V. G. A. Durnin (Institute of Physiology, University of Glasgow): *Energy expenditure.*

The titles of papers are not necessarily in final form.

Dr. D. F. Hollingsworth is the Hon. Programmes Secretary. His address is: c/o Ministry of Agriculture, Fisheries and Food, Great Westminster House, Horseferry Road, London, S.W.1.

PREPARATIONS AND APPLIANCES

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Evans Medical Supplies announce the availability of *Ebimar* and *Ebimar-Al* Tablets for the treatment of peptic ulcers.

Ebimar is a purified and stabilized sulphate polysaccharide derived from seaweed. It inhibits pepsin activity and also has the property of combining with exposed tissue protein, thus preventing enzyme access to the damaged area. Each tablet contains 0.5 gramme of *Ebimar,* representing not less than 1,000 Evans Units of peptic activity.

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The activity of *Ebimar* is unaffected by saliva and it does not inhibit tryptic digestion. Its pepsin-inhibitory action is therefore localized to that part of the gastro-intestinal tract which supports an acid environment and peptic ulcers. Its effect is not diminished in the presence of up to 4% of protein, but higher concentrations lessen the inhibition. *Ebimar* or *Ebimar-Al,* therefore, should be taken before or between meals.

Widespread clinical trials have demonstrated the value of *Ebimar* and *Ebimar-Al* in established cases of peptic ulcer, with complete absence of side effects. Owing to the nature of the condition, some considerable further time must elapse before full evaluation is possible on a long-term basis.

The dosage scheme at present suggested is as follows: three to four tablets to be sucked or chewed, preferably with a glass of water, four or five times a day between meals.

Ebimar tablets are available in containers of 100 and 500 tablets; *Ebimar-Al* in containers of 50 and 250.

Further details may be obtained from Evans Medical Supplies, P.O. Box 6607, Johannesburg.

BOOK REVIEW

COMMUNICABLE DISEASES IN SCHOOL

Communicable Diseases in School: A Survey of Existing Legislation. Offprint from *World Health Organization: International Digest of Health Legislation,* Geneva, Vol. 10, No. 2, 66 pages. 3s. 6d. Pretoria: Van Schaik's Bookstore (Pty.) Ltd., P.O. Box 724.

Communicable diseases are one of the commonest causes of school absenteeism. This survey covers the statutory regulations in force in fifteen countries. The measures prescribed, of which the exclusion from school of sick children and of contacts is paramount, are compared with the recommendations found in medical literature. It is noted that the health authorities tend at present to substitute surveillance for stringent measures. Surveillance presupposes well-organized school health services and the existence of bacteriological laboratories.

The subject matter of the study published in the *Digest* is arranged under the following headings: legislation, exclusion measures, obligations of parents, teaching staff, and health services; measures relating to school staff; and closure of schools. An appendix contains an analysis in tabular form, by country and by disease, of the statutory measures taken in the countries whose legislation is covered by the study.

CORRESPONDENCE

THE DOCTOR'S DUTY IN EPILEPSY

To the Editor: A patient of mine who suffers from epilepsy has now left this area, where he was a poultry farm manager. He was seen a few days ago driving a heavy truck in Johannesburg and is possibly its regular driver.

What shall I do?

H. J. L.

STOP ORDERS AND THE MEDICAL SERVICES PLAN

To the Editor: At the last meeting of the Federal Council of the Medical Association of South Africa held in East London, the Federal Council agreed that the two following Rules as regards Medical Services Plan be accepted:

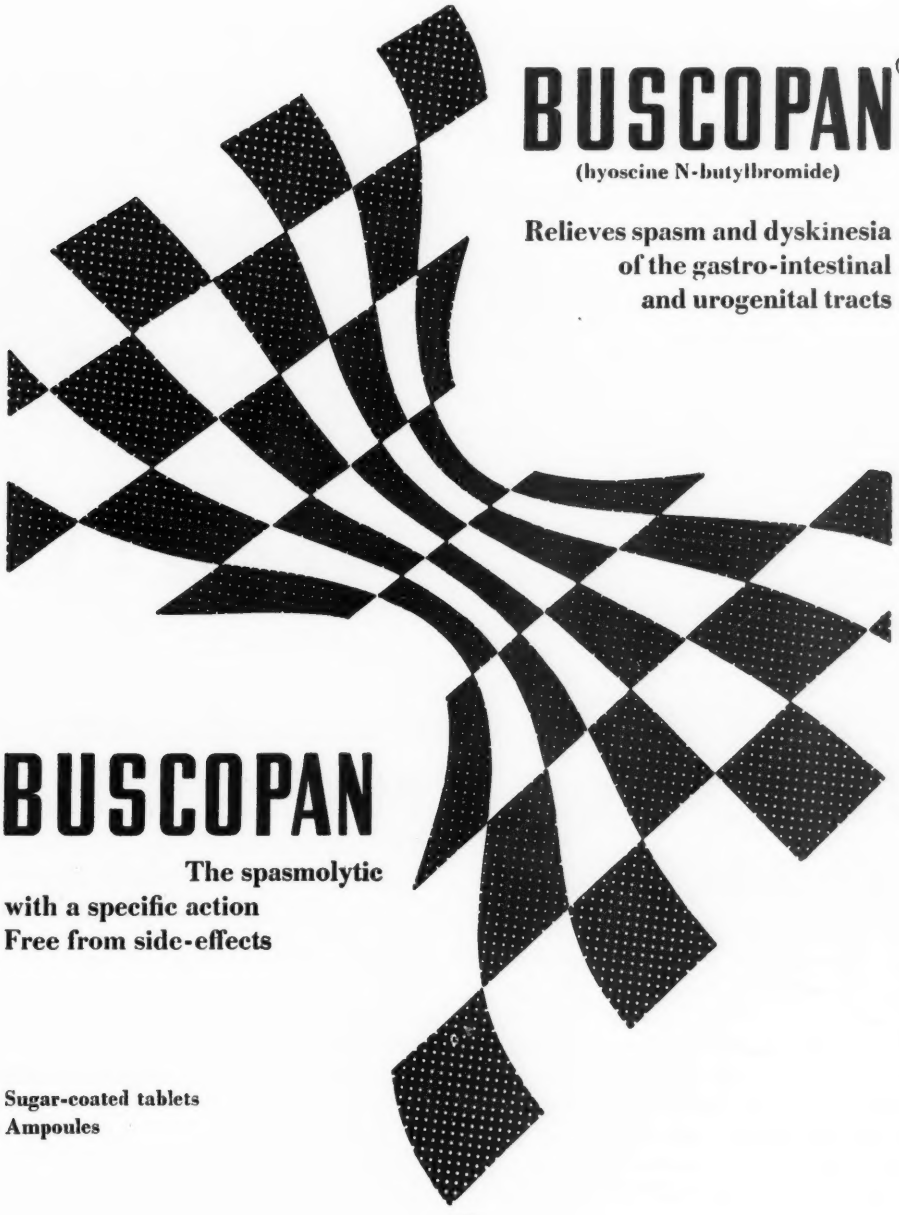
Rule I: 'Where a Subscriber to the Plan or his dependant is obliged to seek medical services in an area where the Plan does not yet operate, the Plan will treat the Medical Practitioner concerned as a Participating Doctor for the purposes of payment, provided that the Medical Practitioner shall have agreed to accept payment from the Plan as a Participating Doctor before rendering services.'

Rule II: 'Subscribing Membersip to Medical Services Plan is open to any registered medical practitioner. Upon acceptance of an application for membership, the medical practitioner and his dependants shall be entitled to all benefits of the Plan in accordance with the terms and conditions set out in the Subscriber's Contract, provided that payment shall either be made annually in advance or otherwise secured to the satisfaction of the Board of Directors of Medical Services Plan.'

With regards to the second Rule, the Board of Directors have decided to accept Stop Order facilities from doctors and their dependants wishing to become Subscribing Members to the Medical Services Plan. The required Stop Order form is available at the Head Office of the Plan, P.O. Box 10314, Johannesburg.

P. J. Parvus,
General Manager.

P.O. Box 10314,
Johannesburg.



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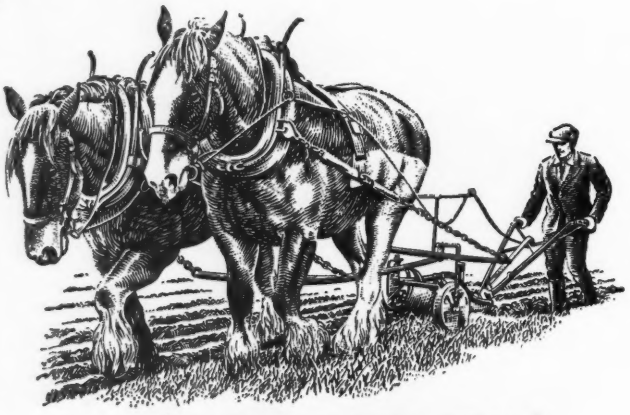

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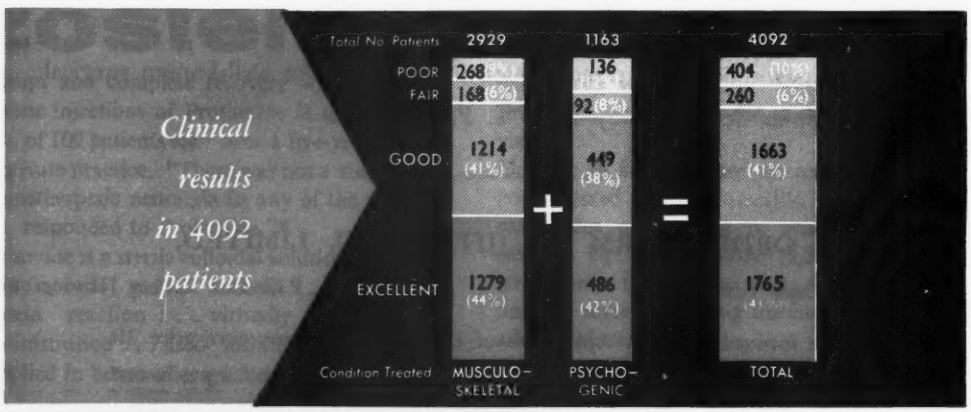
L. *laxare*, to loosen, as the muscles



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Medicus in *The Star*, 8 November 1957.

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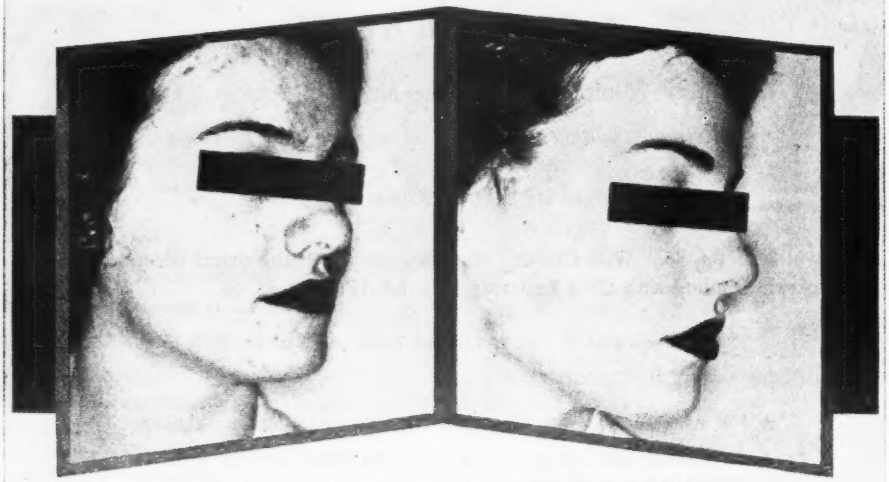

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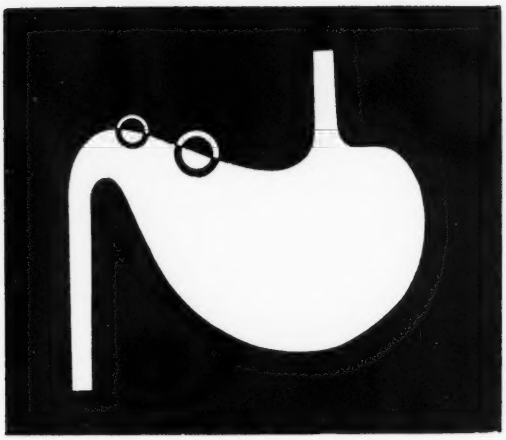

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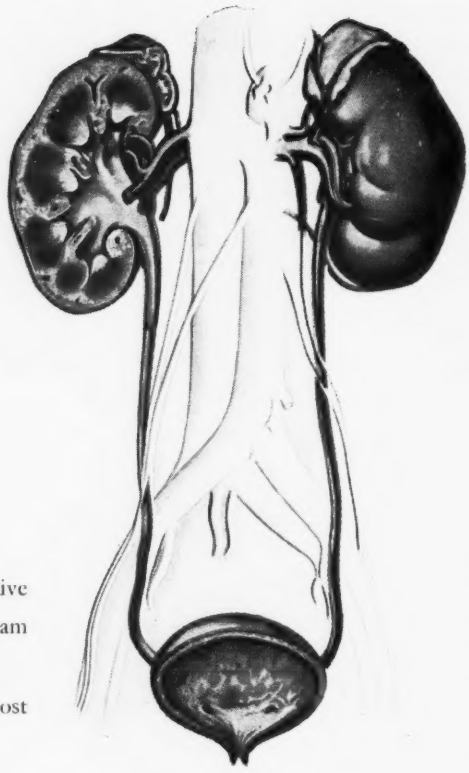

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